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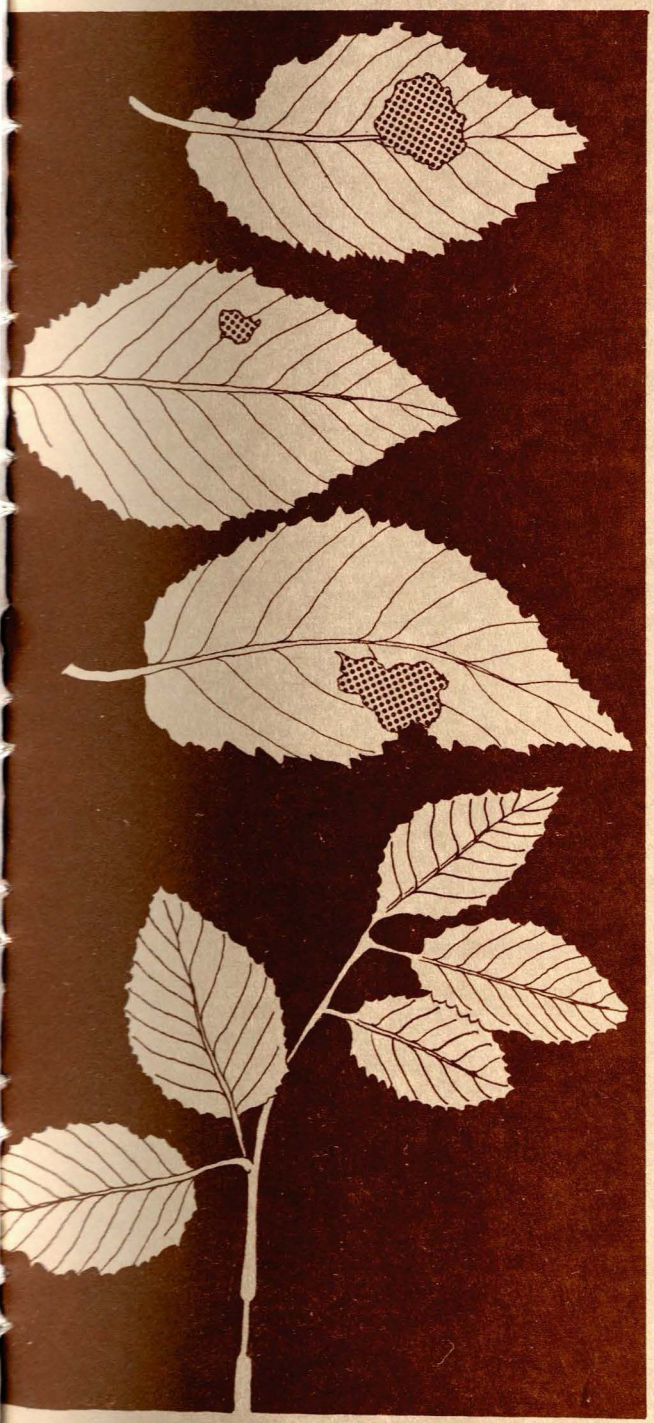
Gnomonia Canker, shoot blight, and leaf spot of YELLOW BIRCH



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GNOMONIA CANKER, SHOOT BLIGHT, AND LEAF SPOT OF YELLOW BIRCH

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For a number of reasons it has been difficult to establish northern hardwood forests in the Great Lakes region that contain a stocking of yellow birch (*Betula alleghaniensis*) similar to that of the climax forests encountered by the early settlers (Tubbs 1969).Several factors that have contributed to the decline of yellow birch are competition with other tree species (Tubbs 1973), animal browsing (Graham 1954, Stoeckeler and Limstrom 1950), and a canker disease caused by *Diaporthe alleghaniensis* Arnold (Arnold 1967, 1970, Kessler 1971, Burton *et al.* 1969). In this paper I report on another fungus, *Gnomonia setacea* (Pers.) Ces. et DeNot., that affects yellow birch regeneration.

MATERIALS AND METHODS

To isolate the fungus, stem cankers were cut into fragments and surface-sterilized in undiluted commercial sodium hypochlorite bleach for 4 to 10 minutes. The canker fragments were then plated on a streptomycin-sucrose-yeast extract (30 ppm - 10 g - 2g/L) agar medium and incubated at 20 C for 1 to 2 weeks. Isolations from fruiting bodies were made by grinding them in sterile water and plating serial dilutions of the resulting spore-mycelial suspensions on the streptomycin-sucrose-yeast extract agar medium.

For germination studies spore masses were added to 100 ml of sucrose-yeast extract solution (10 g:2 g/L) and stirred with a magnetic stirrer for 15 minutes to disperse the spores. Serial dilutions were then made into petri dishes and spore concentrations determined. Percent germination was determined for 200 conidia scattered over at least 10 sites on each dish.

Plants were grown in a growth chamber for 2 months in agricultural perlite to which Knop's nutrient solution was added weekly. After 2 months the plants were inoculated and then watered with KCl solutions to create a moisture stress. Three KCl concentrations were used to create stresses of -5, -8, and -14 bars. Plants were again watered with KCl solutions at 3, 5, and 7 days after inoculation. Ten days after inoculation the KCl solutions were washed from the perlite substrate by repeatedly flushing water through the pots.

Leaves were inoculated by spraying sucrose-yeast extract solution (10 g:2g/L) containing dispersed conidia onto leaf surfaces with a plastic atomizer. Inoculated plants were kept at 100 percent relative humidity for 3 to 5 days and then moved to greenhouse benches for observation.

Stems from 5 to 15 mm in circumference were inoculated by applying drops of conidial suspension to stem wounds that had been made with scalpels, dissecting needles, pruners, long-nosed pliers, dry ice, heated nailsets, or by stem bending. Wounds were covered with moist cotton and aluminum foil. Stems were also inoculated with mycelia. Inoculations were made at internodes on the lower portions of main stems of seedlings. A tangential bark-flap wound was made with a scalpel and a piece of mycelial inoculum, which had been taken from the margin of a 3-day-old fungal colony, was inserted under the flap. Sterile moist cotton was then applied over the wound and covered with aluminum foil.

Field inoculations were also carried out. Each month from May to November 1972, 20 3-year-old yellow birches were inoculated by the scalpel-stem wound method near Hiles, in northern

Wisconsin. The following April, 20 additional trees were inoculated.

To obtain the perfect, perithecial stage, infected leaves with leaf spots were placed on moistened sphagnum in aluminum trays and covered with a 1 to 2 cm layer of moist sphagnum in October 1973. The trays were incubated at 2 C. Each month the leaves were examined for the presence of perithecia.

RESULTS

Distribution

Gnomonia setacea has been isolated from cankers on yellow birch seedlings throughout the commercial range of that tree in Michigan, Minnesota, and Wisconsin. Incidence of main stem girdling cankers in 12 hardwood stands surveyed in Wisconsin and Michigan ranged from 7 to 68 percent (average 28 percent). Recovery rate of the fungus from cankers has ranged from 6 to 69 percent (average 21 percent). The fungus is readily isolated from acervuli on leaf spots during spring and summer. In the fall conidial production from acervuli diminishes and recovery of the fungus from leaves becomes more difficult.

Symptoms

Incipient cankers are confined to the outer bark. As cankers develop, inner bark and even wood may become affected and discolor. Cankers are blackish and may appear sunken in later stages due to a combination of cell collapse and growth of healthy tissues surrounding the cankers (fig. 1, p. 6). In the spring, cankers may girdle the new terminal shoots and cause a shoot blight stage of the disease. Such stems often develop a characteristic "shepherd's crook" bending. Canker's on older wood sometimes completely girdle stems during summer-particularly during drought periods (fig. 2, p. 6). The first indication of imminent summer stem girdling is the development of foliar chlorosis. Chlorosis is then followed by foliar wilting. In some cases cankered stems heal. When this happens, the necrotic tissues are sloughed away and a lenticular gall develops (fig. 3, p. 6;4). Leaves with leaf spot infections generally abscise within 2 weeks. Because of this rapid abscission, it is seldom that more than a few spotted leaves can be found on a tree at any given time. The spots are brown, vary in shape, and are sometimes delimited by major veins (fig. 5, p. 6). Chlorotic zones surround the spots, particularly the larger ones. Size of the spots range from 3 mm to several centimeters.

The shoot blight canker symptoms of yellow birch canker disease caused by *Diaporthe alleghaniensis* Arnold and described by Arnold (1967) cannot be distinguished from those caused by *G. setacea.*

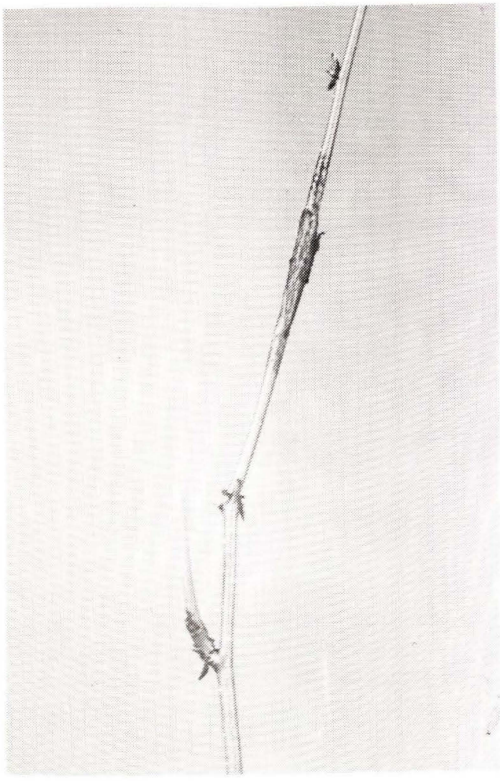


Figure 4.—*Healed canker.*

The Causal Organism

The perithecial perfect stage develops on diseased leaves after the leaves have been shed in the fall. When leaf-spotted leaves collected in July and September, 1973, were incubated at 2C in moist sphagnum or perlite, perithecia were first found on them in early January. The perithecia were generally more numerous on the dorsal than

on the ventral leaf blade surface (fig. 6). However, all leaves were incubated dorsal side up, so this may have affected perithecial distribution.

Figure 6.—*Perithecia that have developed on a fallen leaf.*

Perithecia developed on or close to the leaf spots—more near the spot margins than in the spot center. Fewer perithecia were found on petioles presumably because leaf spots were not contiguous with petioles on many leaves.

The ascocarps develop within the leaf tissues. As the ascocarps enlarge they tend to erupt through the host tissue and become partially exposed. In some cases only the ostiole is visible. As the leaf tissue decays, more and more of the ascocarp is exposed until finally it appears to be resting on the leaf surface.

Perithecia were 166 to 410 µm wide and 154 to 372 µm high and their rostrums ranged from 41 to 455 µm long and from 31 to 74 µm wide. Asci measured 5 to 8 by 20 to 29 µm and were cylindric to clavate with an evanescent stalk and unitunicate wall that was conspicuously thickened at the apex where a refractive ring surrounds the apical pore. The ascus wall was difficult to see even with the aid of stains or phase contrast microscopy (fig. 7). Ascospores were 1.2 and 2.4 µm wide by 8.4 to 14.6 µm long, two-celled, hyaline with thin-walled appendages at the ends, cylindrical, tapered slightly at the ends, and were straight to slightly curved (fig. 8).

Figure 7.—*Asci.*

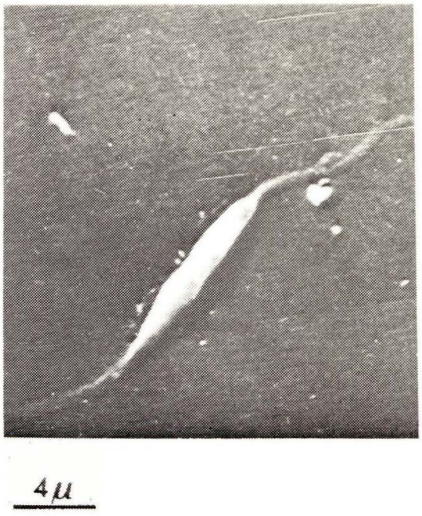


Figure 8.—*Ascospore.*

The perfect stage is *Gnomonia setacea* (Pers.) Ces et DeNot (table 1). This identification is based largely on the size of the perithecia, asci, and ascospores and on the presence of evanescent appendages at both ends of ascospores. *Gnomonia setacea* and *G. betulina* Vleugel, are the only *Gnomonia* species that have been reported on *Betula* spp. that have small ascospores bearing evanescent appendages. Ascospores of *G. betulina,* however, are much larger (Vleugel 1917) than those of *G. setacea.*

The acervulus imperfect stage develops on leaf spots (fig. 9, p. 7) and on stem cankers during the growing season. On the leaf tissue the acervuli develop subcuticularly (fig. 10). Phialophores are

Table 1.—*Comparison of the perithecial stage of yellow birch fungus with* Gnomonia setacea *of other authors*

Authority	Perithecial width	Asci	Ascospores	Hosts
		µm		
		G. SETACEA		
Kessler	166-410	20-29 by 5-8	8.4-14.6 by 1.8-2.4	*Betula alleghaniensis*
Saccardo 1882	150-300	30-40 by 6-9	14-15 by 1.5-2	*Quercus, castanea, Acer, Crataegus Betula, Alnus, Corylus*
Ellis & Everhart 1892	200-300	30-40 by 6-9	12-16 by 1.5-2	*Quercus, Castanea,* and other hardwoods
Rabenhorst 1887	200-300	30-40 by 6-9	14-16 by 1.5-2	Various tree and shrub species
Muller & von Arx 1962	200-400	30-40 by 6-9	14-16 by 1.5-2	Various tree and shrub species
		G. SETACEA f. *ALNI*		
Vleugel 1911	--	23-31 by 5-10 mature 57 by 13	10-13 by 1.5-2	*Alnus incana, A. borealis*
Klebahn 1918	140-180	25-30 by 6	11-13 by 1.5-1.7	
		G. SETACEA f. *BETULAE*		
Vleugel 1911	--	26-29 by 6-10	14 by 2.5	*Betula* sp.

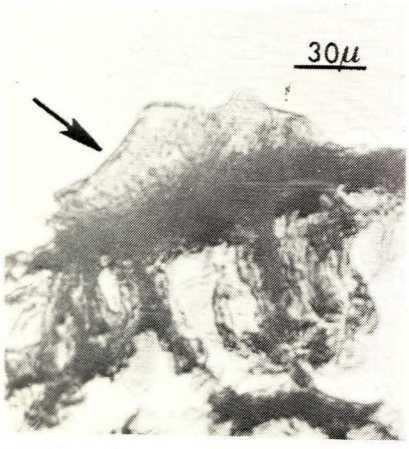


Figure 10.—*Cross-section of acervulus on leaf with intact host cuticle (arrow).*

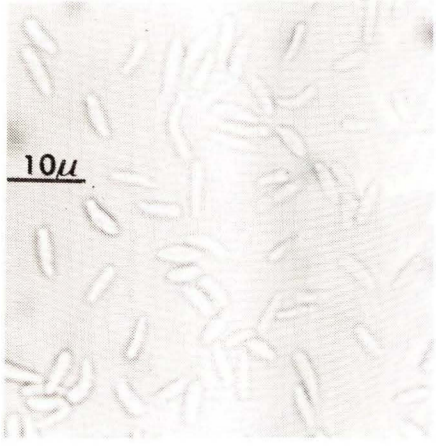


Figure 11.—*Swollen conidia prior to germination. Notice different shapes.*

densely crowded. The conidia vary in shape and range from rod-shaped to allantoid (fig. 11). Prior to germination conidia imbibe water and increase their size markedly.

In culture the fungus develops zonate colonies with a cottony-feathery aerial mycelium (fig. 12, p. 7). Acervuli are produced abundantly in culture, particularly within the agar medium (fig. 13, p. 7).

Disease Cycle

Conidia require nutrients to germinate. Sucrose-yeast extract solutions, in particular, supported good germination. When nutrients present in sucrose-yeast extract were tested individually, results were extremely variable. In some experiments the addition of sucrose to distilled water increased the percentage of germination although its addition had little effect in other experiments. Nitrogen sources, in particular asparagine, enhanced germination. Conidia sometimes swelled and developed slight germ tube-like protuberances in distilled water but usually were unable to develop further.

At temperatures of 7 C or less, more than 24 hours were required for conidial germ tubes to reach a length capable of penetrating host cells (table 2.). After 24 hours, growth was limited at 13 degrees or lower, moderate at 16 degrees, and extensive at temperatures from 19 to 30 C.

Optimal temperatures for early germ tube extension were 25 to 28 C. After 48 hours growth was still limited at 7 degrees or lower, moderate at 12 degrees, and extensive at temperatures from 16 to 30 C (fig. 14). Germ tubes developing at 30 C often coiled or curled abnormally. Little or no germ tube development occurred at 32 C.

Table 2.—*Development of conidial germ tubes at temperatures from 1 to 30 C*

Temperature (C)	Time (hours) 24	48	108
		µm	
1	0	3	11
7	0	7	>200
12	3	13	>400
13	5	165	>600
16	13	340	>600
19	29	>600	>600
22	55	>600	>600
25	96	>600	>600
28	83	>600	>600
30	26	113	>600

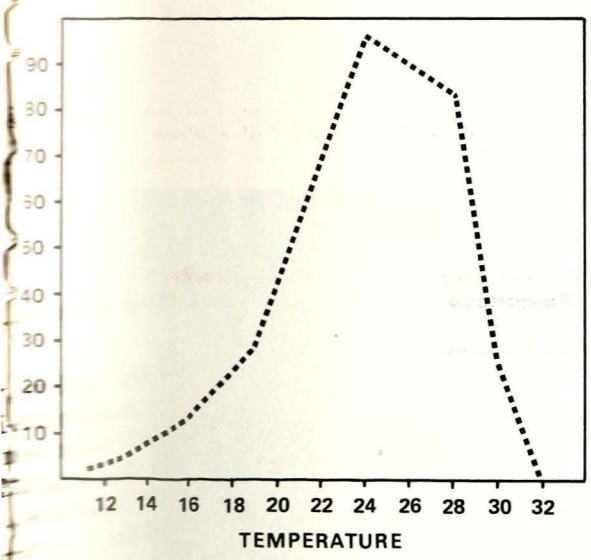


Figure 14.—*Relation of conidial germ tube length to temperature. Incubation time 24 hours.*

A self-inhibitor severely restricts spore germination at conidial concentrations greater than 50 spores/mm^3 (fig. 15). Spores did not germinate at high percentage rates unless they occurred in concentrations of 20/mm^3 or less. At conidial concentrations of 800 spores or more per mm^3, spores failed to germinate. Attempts to wash the

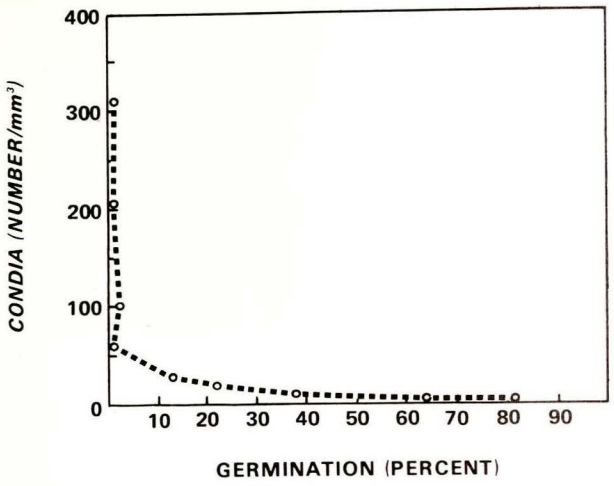


Figure 15.—*The effect of conidial concentration on germination.*

germination inhibitor off the spores were unsuccessful.

Leaves and petioles were more susceptible to conidial infection than were buds and stems. Buds were the most resistant part of the plant (10 percent successful infections compared to 98 for leaves, 75 for petioles, and 25 for stems). After 10 days leaf spot size on inoculated apical leaves averaged 10 by 8 mm. Canker development was greater at nodes than at internodes; 5 of 15 inoculations at nodes resulted in stem girdling cankers and comparable inoculations at internodes produced no girdling cankers.

All wounds that ruptured the bark could be infected (see tabulation below).

Wound type	Percent stem circumference affected: Inoculated wound (A)	Control wound (B)	Pathogen affect (A-B)
Needle	50	13	37
Scalpel scrape	33	0	33
Heated nailset	52	50	2
Knife cut	49	21	28
Dry ice and needle	39	28	11
Dry ice and scalpel scrape	56	39	17
Dry ice	36	37	-1
Dry ice and knife cut	66	57	9
Nailset	49	40	9
Stem bending	50	0	50

Size of wound was not as important as type of wound; needle wounds developed comparatively much larger cankers than tangential knife cuts. When an adjustment is made for wound size, canker development arising from the invasion of *Gnomonia setacea* can be determined and compared for different types of stem wounding.

Figure 1.—*Canker on young seedling (dark area).*

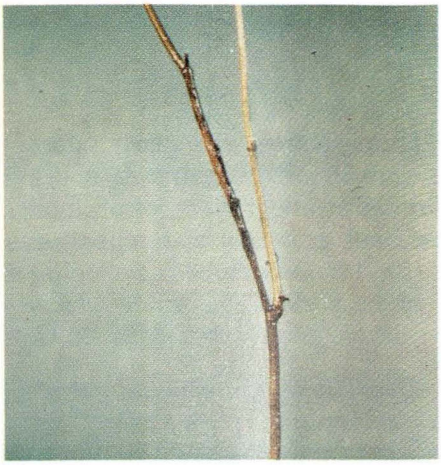


Figure 3.—*Healing canker sloughing off outer bark.*

Figure 2.—*Effects of stem girdling by a canker during a summer drought.*

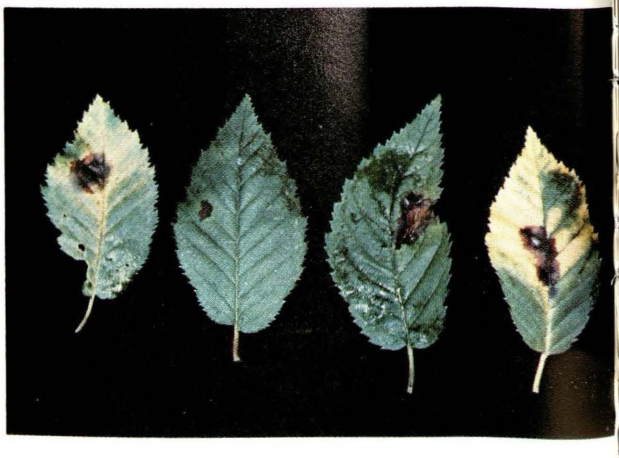


Figure 5.—*Leaf spots.*

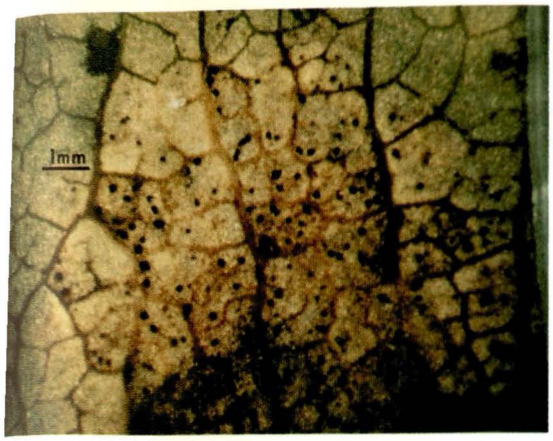


Figure 9.—*Acervuli on leaf spot.*

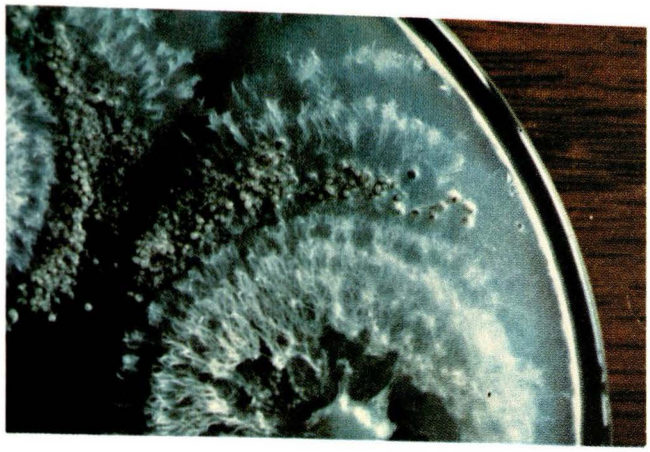


Figure 13.—*Masses of conidia produced in acervuli that developed in agar plate culture.*

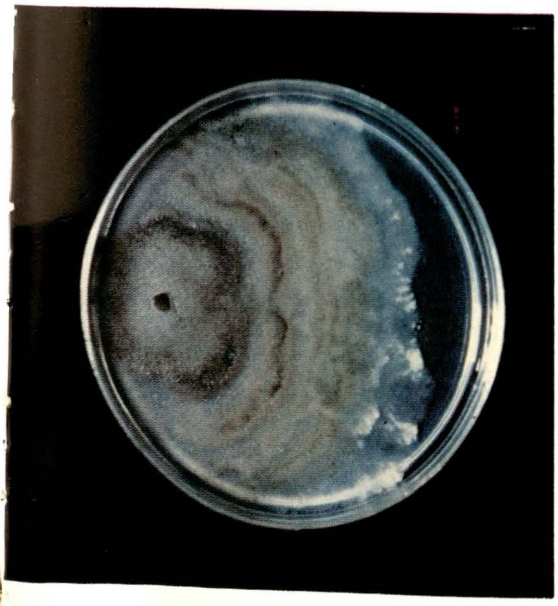


Figure 12.—*Growth of typical isolate of* Gnomonia setacea *on sucrose-yeast extract agar medium.*

Wounds that did not rupture the bark, such as those made by dry ice or hot nailset applications to stems, did not become colonized, even though more than a third of a stem's circumference was injured in creating such wounds. Some wounds, such as dry ice injury followed by a knife cut into the wounded area, developed into large cankers. When the correction was applied for wounding injury, the contribution of *G. setacea* to the enlargement for such wounds was low. The wounds that enabled *G. setacea* to influence canker size the most were stem needle wounds and stem bending. If bent stems were inoculated, about half of them became girdled at the point of bending (fig. 16, p. 9). Stems that were clipped and inoculated where they were cut off died back to the first live bud. Uninoculated clipped stems did not die back.

Fresh stem wounds were more susceptible to infection than older wounds (fig. 17, p. 9). Stem wounds gradually became less susceptible in the week following wounding.

Stem age was not as important as the degree of stem lignification in influencing susceptibility. Succulent stems were more susceptible to cankering than lignified ones. When trees were inoculated at three stem locations, all inoculations of

Figure 18.—*Inoculation on current season's growth (upper arrow) resulted in stem girdling and wilting of foliage. Moderate-sized canker (upper inset) developed after inoculation of 2-year-old portion of stem. Small canker (lower inset) developed after inoculation of 5-year-old stem.*

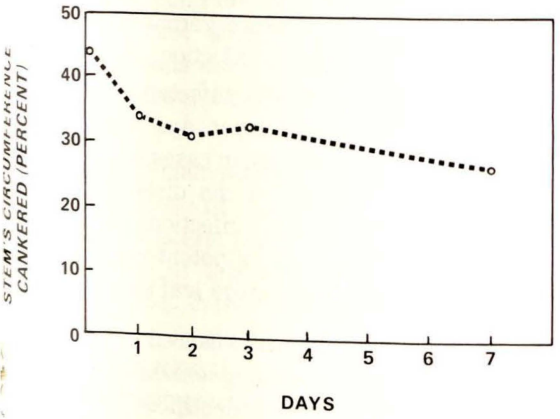
Figure 22.—*Ascospore inoculation of petiole scar. Note canker development (arrow).*

succulent current season's terminal growth resulted in stem girdling cankers, basal stem inoculations (stem age 5 years) resulted in small cankers, and inoculations of 2-year-old stems resulted in moderate-sized cankers (fig. 18, above).

Creating moisture stress conditions in inoculated plants irrigated with potassium chloride–Knop's solutions increased the size of basal stem cankers (see tabulation below).

Osmotic pressure	Stem circumference cankered
(Bars)	(Percent)
0	36
-5	44
-8	60
-14	72

gure 16.—*Plants on left were inoculated with conidia after their stems were bent. Plants on the right had their stems bent but were not inoculated.*



igure 17.—*Effect of age of wound on canker development.*

Foliar moisture stress symptoms also developed. All foliage on -14 bar treated trees (severe moisture stress) and most of the foliage of the -8 bar treated trees became brown. Foliage of -5 bar treated trees (moderate moisture stress) was discolored at the leaf margins.

Gnomonia setacea grows best at temperatures from 20 to 26 C but it is capable of slow growth at temperatures of 1 to 10 C on a sucrose-yeast extract agar medium. At a temperature of 32 C growth is restricted and at 34 C growth does not occur (fig. 19). When stems are inoculated with the fungus in the spring, more canker development occurs at moderate temperatures than at low temperatures (table 3.). When inoculations were conducted on dormant birch during mid-winter, however, plants kept chilled at 2.5 C developed larger cankers than did inoculated plants transferred immediately to a greenhouse environment (fig. 20).

Monthly inoculations of yellow birch nursery stock planted in a northern hardwood shelterwood forest situation in northern Wisconsin revealed that the stems were most susceptible during June (fig. 21). Susceptibility decreased as the growing season progressed and by October the stems were resistant. Comparative inoculations made with *Diaporthe alleghaniensis* indicated that the two fungi followed a similar seasonal susceptibility pattern.

The most common sources of conidial inoculum are acervuli that develop on leaf spots during spring and summer. Although leaf spotting of sawtimber-sized yellow birch is generally not severe, the overstory position of these birch assure good conidial spread from their spotted leaves to smaller yellow birch beneath them. Acervuli also develop on cankers and blighted shoots, and conidia from these sources may contribute to the numerous multiple infections encountered on stems of natural seedlings. Conidial production from acervuli on leaf spots diminishes in the fall and by late October has virtually ceased. Leaves with leaf spots soon abscise and fall to the ground. Perithecia develop on fallen leaves and completely formed ones (but without asci) have been found as early as the end of October. When perithicia-containing leaf spots were incubated overwinter at 2 C on moistened sphagnum or perlite, mature ascospores did not form until April.

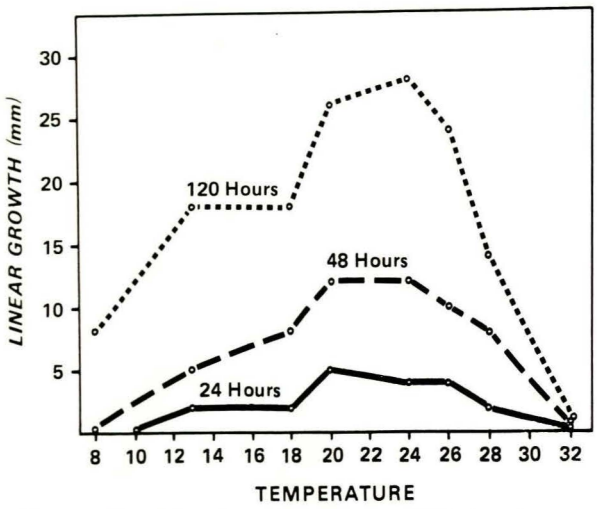


Figure 19—*Relation of linear growth* in vitro *to temperature.*

Table 3.—*Effect of chilling inoculated dormant stock on the development of cankers in June*
(In percent of stem circumference cankered)

Inoculum incubation temperature (C)	Plant treatment after inoculation: Planted and transferred to greenhouse	4 weeks at 5 C	4 weeks at 2.5 C
5	45	26	28
15	48	30	28
25	58	33	32
Average	50	30	29

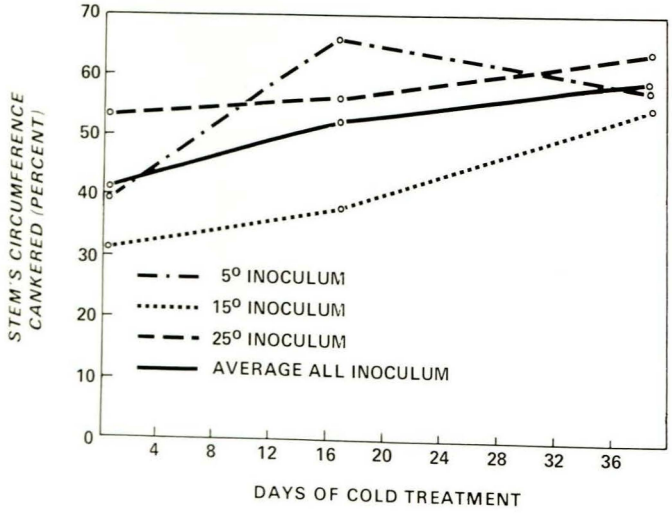


Figure 20—*Effect of length of chilling (2.5 C) dormant yellow birch on the development of stem cankers arising from inoculation in January.*

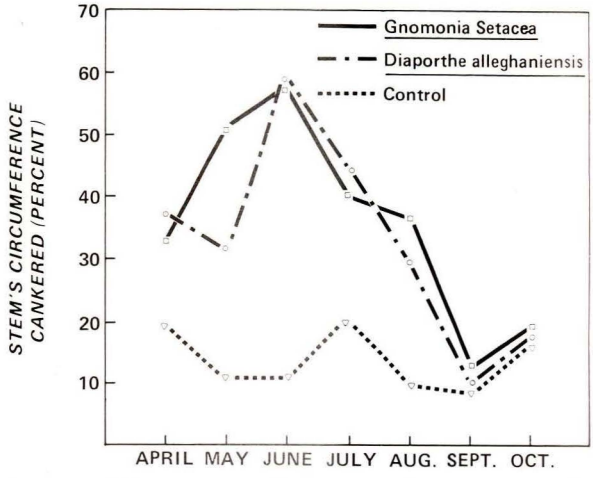


Figure 21.—*Seasonal susceptibility of yellow birch to cankering by* Gnomonia setacea *and* Diaporthe alleghaniensis.

DISCUSSION

The link between the leaf spot phase of this disease complex and the canker and shoot blight phases was not established until the fall of 1973. Prior to that time the primary source of spring and fall inoculum was thought to be conidia. With the discovery of the ascospore stage it now appears that ascospores are primary inoculum for stem cankers that appear in the spring (fig. 22, p. 8).

Self-inhibition of conidial germination serves as a mechanism to limit germination to situations where conidial concentrations are low. In addition, the conidia require nutrients to germinate. Wound and leaf surfaces provide adequate nutrients to support conidial germination and subsequent host infection. Self-inhibitors and nutrient requirements thus tend to enhance germination success in situations favorable to infection.

When leaves become infected, they soon develop an abscission layer and drop to the ground. This happens even in cases where a small infection spot develops at the distal end of the leaf. Apparently fungal infection of the leaf initiates the production of a potent toxin which in some way initiates premature leaf abscission.

Cankers and shoot blights caused by *Gnomonia setacea* and *Diaporthe alleghaniensis* cannot be visually distinguished. Indentification requires examination of fruiting bodies or isolation to obtain the causal agents in pure culture (Kessler 1976). In a few cases both fungi have been isolated

rom the same canker. Artificial inoculations of stem wounds with both fungi indicated, however, hat the two fungi have little synergistic effect.

Although Vleugel (1911) described a form *betulae* of *G. setacea,* it was based solely on occurrence on *Betula* sp. (table 1). Until host range studies have been conducted with isolates collected from several hosts, the form *betulae* designation should not be used.

The name of the imperfect stage of this fungus s *Cylindrosporella microsperma* (Peck) Petr. (von Arx 1970). It was first described by Peck (1883) as *Septoria microsperma* (Peck). However, because he conidia were not septate, *Septoria* was an ncorrect designation of the imperfect stage. Davis 1915), therefore, renamed it *Sacidium microspermum* (Peck). The same year Saccardo (1915), vithout mentioning either *Septoria microsperma* r *Sacidium microspermum,* named the imperfect stage *Gloeosporium betulae-luteae* Sacc. et Dearn. The genus *Sacidium,* based on an mperfectly described type species, is not valid. This led Petrak (1924) to rename the fungus *Cylindrosporella microsperma* (Peck) Petr. Petrak's name for the imperfect stage was upheld by von Arx in 1970. Von Arx considers the genus *Gloeosporium* to be too heterogenous to be valid any longer. So if his concept of *Gloeosporium* is followed, Saccardo and Dearness' name for the fungus, *G. betula-eluteae* should not be used. I agree with von Arx and, therefore, have used the name *Cylindrosporella microsperma* (Peck) Petrak for the imperfect stage.

Buds were more resistant to infection than were leaves, stems and petioles. In nature, cankers often appear around dormant buds in the spring. However, because artificial inoculation studies revealed that buds are comparatively resistant to infection, the natural cankers at buds may arise from petiole or petiole scar infections.

Cankering of main stems often results in stem girdling, a loss of apical dominance, and proliferation of lateral branches (figs. 23 and 24). "Bushy" birch of this type tend to lose their dominant position in the reproductive layer of the forest and are frequently overtopped by surrounding vegetation. In some cases stems healed themselves by sloughing off cankered bark tissue, which resulted in a lenticular-shaped gall (fig. 4). Such galls were common on birch seedlings in natural stands.

Figure 23.—*One-year-old dormant birch seedlings. Lateral branches developed after canker girdled main stems.*

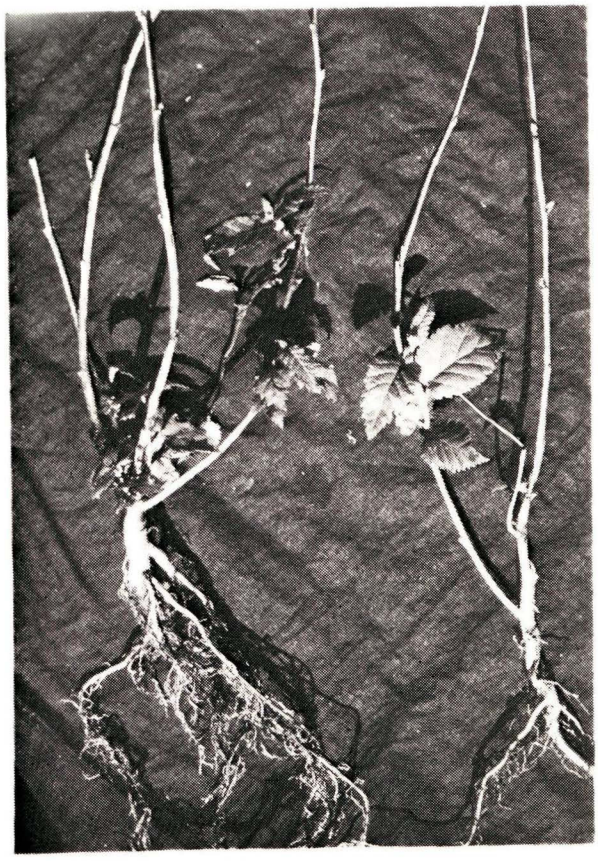


Figure 24—*Three-year-old birch. Lateral branches developed after canker girdled main stems.*

FOREST MANAGEMENT IMPLICATIONS

Cankering and shoot blight are most serious on yellow birch seedlings with stem diameters less than 2 cm. By maintaining optimal growing conditions, the years of small seedling susceptibility can be reduced to 3 to 5 years. Seedlings growing on poor sites or subjected to unfavorable growing conditions such as drought or competition are much more likely to become infected and will remain susceptible for many years.

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Describes a canker, shoot blight, and leaf spot disease of yellow birch seedlings in the northern Great Lakes region and tells how and when trees become infected by the fungal causal agent, *Gnomonia setacea.*

OXFORD: 443.2:176.1 *Betula alleghaniensis.* KEY WORDS: *Gnomonia setacea, Cylindrosporella microsperma, Diaporthe alleghaniensis, Betula alleghaniensis.*

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Nature is beautiful...leave only your footprints.